

domized clinical study to evaluate this hypothesis is justifiable.

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Combined mitral valve repair and the Cox maze procedure for mitral valve prolapse and regurgitation and associated atrial fibrillation

To the Editor:

I read with great interest the report by Handa and associates¹ on the early and intermediate term results of combined mitral valve repair and the Cox maze procedure for patients with severe mitral regurgitation and associated atrial fibrillation. It is gratifying to note that at the Mayo Clinic the addition of the Cox maze procedure to mitral valve repair not only is safe and effective but also decreases late complications.

One of the serious complications of mitral valve prolapse, which is otherwise a common² and benign³ condition, is mitral regurgitation.^{4,5} In fact, the commonest cause of mitral regurgitation in the United States is mitral valve prolapse.^{5,6} Mitral regurgitation in mitral valve prolapse may become progressive in some patients.⁵ One of the serious complications of progressive mitral regurgitation is atrial fibrillation, which usually persists even after successful corrective surgery of the mitral valve and often recurs after pharmacologic or electric cardioversion. Although postoperative atrial fibrillation can be successfully managed by antiarrhythmic drugs and long-term anticoagulant therapy to prevent thromboembolism, these therapeutic modalities are not without side-effects, torsades de pointes and bleeding, respectively. It is gratifying that the adjunctive Cox maze procedure at the Mayo Clinic increased the restoration of sinus rhythm to 82% of their patients.

Therefore, as the authors concluded, "Although the addition of the Cox maze procedure lengthens hospital stay in patients having mitral valve repair, overall medical costs may be decreased by reducing the need for long-term anticoagulation, as well as costs associated with stroke and bleeding complications."¹

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Should the bidirectional Glenn procedure be better performed through the support of cardiopulmonary bypass?

To the Editor:

We read with great interest the report of Jahangiri and associates¹ and the commentary by Jonas on the technique of bidirectional Glenn shunt through a thoracotomy without the use of cardiopulmonary bypass (CPB). We agree that clamping the superior vena cava (SVC) without decompressing the internal jugular system exposes the brain to the effects of reduced cerebral perfusion pressure. We have used electrophysiological indices of cerebral function (electroencephalography or evoked potentials) and transcranial Doppler during bidirectional Glenn shunts in patients with pulmonary atresia without support (n = 2) or through the use of CPB (n = 4). During clamping of the SVC without CPB, major reductions (>50%) in the diastolic, mean, and peak systolic blood flow velocities of the middle cerebral artery were identified, which were followed by mild electrocortical alterations as indicated by longer latencies of the cortically generated evoked potentials.² In contrast, this situation did not occur or was minimal in those cases in which clamping of the SVC was done with the support of CPB. We have learned that Doppler flow changes and electrocortical alterations may be expected as a result of acute SVC hypertension if the internal jugular venous system is not decompressed.³ In one of our cases without CPB, an intraoperative shunt into the SVC was indicated due to the presence of extremely low flow velocities and electrocortical alterations during SVC clamping. In this case, while the SVC shunt was patent, flow velocities were maintained and the latencies of evoked potentials returned to preclamping levels.⁴ In our institution, we routinely use CPB and intraoperative brain function monitoring by transcranial Doppler, near-infrared spectrophotometry, and electroencephalography for children undergoing these procedures.³ A note of caution should be made that the reported absence of gross neurologic deficits in Jahangiri and colleagues' cases is not an indicator that the brain is free of potential alterations during SVC clamping without CPB. Furthermore, under conditions of normothermic ischemia, the brain does not receive the protective benefit of hypothermia.⁵ Without brain protection, even short intervals of low cerebral perfusion could generate minor or subclinical deficits that might be detectable only through detailed cognitive testing. We agree with Jonas' comments that the demonstration of safety of this procedure will rest on the careful

documentation of the neurodevelopmental and cognitive outcome of these patients.

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Reply to the Editor:

We appreciate the comments of Rodriguez and colleagues. Although the patient was not actively cooled, a low ambient temperature allowed the patient's body temperature to initially drift to around 33°C, necessitating active rewarming at the end of the operation. It is known that moderate to mild (26°C-35°C) hypoperfusion can attenuate the so-called excitotoxic cascade associated with cerebral hypoperfusion. Similarly, there is a fairly steep exponential fall in the cerebral metabolic rate for oxygen, with temperatures ranging from normothermia to 35°C. These facts, taken together with the short superior vena cava clamp time, should minimize the risk of neurologic damage.

We will endeavor to supplement future attempts at performing the bidirectional Glenn procedure without bypass with near-infrared spectroscopy of transcranial Doppler assessment of cerebral blood flow. In addition, we agree with the authors that demonstrating the safety of this operation will need detailed assessment of the patients by neurologic and psychometric testing.

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Preimplantation retrograde pneumoplegia in clinical lung transplantation

To the Editor:

I read with interest the article by Venuta and associates¹ concerning 14 consecutive patients who underwent lung transplantation. The first patients, group I, had antegrade pneumoplegia with Euro-Collins solution preceded by pulmonary arterial flushing with alprostadil (prostaglandin E₁). In group II, additional retrograde flushing was given via the pulmonary veins just before implantation. The authors concluded that retrograde flushing is not detrimental and helps to improve early graft function. The chest x-ray score was significantly better in group II, intubation time was less, and the ratio of arterial PO₂ to inspired oxygen fraction was improved, although the differences did not reach statistical significance.

My colleagues and I² were the first group to use retrograde pneumoplegia in clinical transplantation. However, we did not use antegrade pneumoplegia in those patients, nor did we use alprostadil. The retrograde pneumoplegic solution was given via the left atrium and the flush solution was vented through an incision in the pulmonary artery. This was done immediately after the aorta was crossclamped and cardioplegia established. The lung almost seemed to light up as the retrograde infusion started. Retrograde pneumoplegia was associated with excellent oxygenation, which remains the most sensitive indicator of the adequacy of lung preservation, as well as the lack of any reimplantation response postoperatively, a finding also shown by the current study. What Venuta's study really demonstrates is that retrograde pneumoplegia may confer additional benefits to lung preservation compared with those of antegrade pneumoplegia alone.

Two criticisms can be leveled at this study. First, retrograde pneumoplegia was given after harvesting of the donor lung, not at the time of the harvesting. This point has already been addressed, and the current practice of the group is to give the retrograde perfusion solution immediately after the antegrade perfusion is finished. The second criticism is that pneumoplegic solution was given individually into each pulmonary vein. This method is at variance with our technique, in which the pneumoplegic solution was given into the left atrium and therefore had a uniform distribution through both lungs, something that cannot be guaranteed if individual veins are perfused separately.

I do congratulate Venuta and his colleagues for highlighting the potential advantage of retrograde pulmonary perfusion in clinical lung transplantation.

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